Selective Deficiency of Immunoglobulin A (IgA) And Autoimmunity

A significant correlation between the occurrence of autoimmune hypersensitivity disease and selective deficiency of IgA has been established. The apparent explanation of the correlation is that IgA deficiency allows selective absorption of antigens via the gastrointestinal and respiratory tract. These antigens may provoke an immune response that exhibits cross reaction with host tissues. Antitissue antibodies are common in selective IgA deficiency as are hypersensitivity diseases such as lupus erythematosus.

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Detection of Immune Complexes in Human Serum by Precipitin Reaction With C1q* Component of Complement

Immune complexes formed by antigen-antibody reactions within the circulation can be detected by their reactivity with C1q in gel diffusion producing a precipitin line at the point of interaction. This technique should prove quite useful in the clinical diagnosis of immune complex disease.

C1q is not yet commercially available and is somewhat difficult to prepare in the laboratory, thus currently limiting application of this technique. The method has been successful in detecting immune complexes in patients with lupus erythematosus and in joint fluids from rheumatoid arthritis where the serum complement was low. EDTA enhances the precipitin reaction. Aggregated gamma globulin will also give this precipitin reaction.

*First component of complement.

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C3* Lytic Factor in Nephritis

A factor responsible for lowering complement levels has been identified in the serum of patients with progressive hypocomplementemic glomerulonephritis. This factor acts on complement by combining with a beta globulin cofactor to produce an enzymatic complex that degrades C3 by cleavage to smaller fragments. It is similar in many ways to a factor isolated from the venom of the cobra (Naja naja) which also activates a serum beta globulin to enzymatically cleave C3. None of these factors or cofactors are identifiable with complement components. The lytic factor appears to be responsible for the lower serum complement. Corticosteroid therapy of the patients with hypocomplementemic nephritis results in disappearance of the lytic factor and return of normal complement levels.

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*Third component of complement.

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